LISTING OF CLAIMS:

This listing of claims is provided for the convenience of the Office and does not include any further amendments.

1-31. (Canceled).

32. (Original) A method of modulating the transport in a neuron of a tetanus toxin or a fusion protein comprising a fragment C of the tetanus toxin, wherein the method comprises administering to the neuron a TrkB receptor agonist or a TrkB receptor antagonist in an amount sufficient to modulate the neuronal transport of the tetanus toxin or the fusion protein.

33. (Original) The method according to claim 32, wherein the TrkB receptor agonist is administered, thereby increasing the internalization of the tetanus toxin or fusion protein at a neuromuscular junction.

34. (Original) The method according to claim 33, wherein the TrkB receptor agonist is a neurotrophic factor that activates a TrkB receptor.

35. (Original) The method according to claim 34, wherein the neurotrophic factor is a Brain Derivated Neurotrophic Factor or a Neurotrophin 4.

36. (Original) The method according to claim 33, wherein the TrkB receptor agonist is an antibody that binds to a TrkB receptor, thereby activating the TrkB receptor.

37. (Previously Presented) The method according to claim 35, wherein the internalization of the fusion protein at the neuromuscular junction is increased.

38. (Original) The method according to claim 32, wherein the TrkB receptor antagonist is administered, thereby decreasing the internalization of the tetanus toxin or fusion protein at a neuromuscular junction.

39. (Original) The method according to claim 38, wherein the TrkB receptor antagonist is an antibody that binds to a TrkB receptor agonist, thereby reducing activation of a TrkB receptor.

40. (Original) The method according to claim 39, wherein the TrkB receptor agonist is a neurotrophic factor that activates a TrkB receptor.

41. (Original) The method according to claim 40, wherein the neurotrophic factor is a Brain Derivated Neurotrophic Factor or a Neurotrophin 4.

42. (Previously Presented) The method according to claim 41, wherein the internalization of the tetanus toxin at the neuromuscular junction is decreased.

43. (Original) The method according to claim 40, wherein the neurotrophic factor is administered concurrently with the fusion protein.

44. (Original) A method of modulating the transport in a neuron of a tetanus toxin or a fusion protein comprising a fragment C of the tetanus toxin, wherein the method comprises administering to the neuron a GFRα/cRET receptor agonist or a GFRα/cRET receptor antagonist in an amount sufficient to modulate the neuronal transport of the tetanus toxin or the fusion protein.

45. (Original) The method according to claim 44, wherein the GFRα/cRET receptor agonist is administered, thereby increasing the internalization of the tetanus toxin or fusion protein at a neuromuscular junction.

46. (Original) The method according to claim 45, wherein the GFRα/cRET receptor agonist is a neurotrophic factor that activates a GFRα/cRET receptor.

47. (Original) The method according to claim 46, wherein the neurotrophic factor is a Glial-Derived Neurotrophic Factor.

48. (Original) The method according to claim 44, wherein the GFRα/cRET receptor agonist is an antibody that binds to a GFRα/cRET receptor, thereby activating the GFRα/cRET receptor.

49. (Previously Presented) The method according to claim 46, wherein the internalization of the fusion protein at the neuromuscular junction is increased.

50. (Original) The method according to claim 44, wherein the GFRα/cRET receptor antagonist is administered, thereby decreasing the internalization of the tetanus toxin or fusion protein at a neuromuscular junction.

51. (Original) The method according to claim 50, wherein the GFRα/cRET receptor antagonist is an antibody that binds to a GFRα/cRET receptor agonist, thereby reducing activation of a GFRα/cRET receptor.

52. (Original) The method according to claim 51, wherein the GFRα/cRET receptor agonist is a neurotrophic factor that activates a GFRα/cRET receptor.

53. (Original) The method according to claim 52, wherein the neurotrophic factor is a Glial-Derived Neurotrophic Factor.

54. (Original) The method of claim 53, wherein the internalization of the tetanus toxin at the neuromuscular junction is decreased.

55. (Original) The method according to claim 47, wherein the neurotrophic factor is administered concurrently with the fusion protein.

56. (Original) A composition, comprising a TrkB receptor agonist and a fusion protein comprising a fragment C of the tetanus toxin fused to a second protein.

57. (Original) The composition according to claim 56, wherein, the TrkB receptor antagonist is a neurotrophic factor that activates a TrkB receptor.

58. (Original) The composition according to claim 57, wherein the neurotrophic factor is a Brain Derivated Neurotrophic Factor or a Neurotrophin 4.

59. (Original) A composition, comprising a GFRα/cRET receptor agonist and a fusion protein comprising a fragment C of the tetanus toxin fused to a second protein.

60. (Original) The composition according to claim 59, wherein, the GFRα/cRET receptor antagonist is a neurotrophic factor that activates a GFRα/cRET receptor.

61. (Original) The composition according to claim 60, wherein the neurotrophic factor is Glial-Derived Neurotrophic Factor.

62. (Original) A method of detecting an effect of a compound on neuronal transport, comprising administering to a neuron the compound and a fusion protein comprising a fragment C of the tetanus toxin fused to a second protein, wherein the second protein is encoded by a reporter gene, and detecting the second protein to determine the effect of the compound on neuronal transport.

63. (Previously Presented) The method according to claim 62, wherein the compound is a neurotrophic factor.

64. (Original) A method of screening for a compound that reduces or prevents transport of a tetanus toxin in a neuron, comprising administering to the neuron the compound and a fusion protein comprising a fragment C of the tetanus toxin fused to a

second protein, wherein the second protein is encoded by a reporter gene, detecting the second protein, and selecting the compound that reduces or prevents the neuronal transport of the fusion protein.

65. (Original) The method according to claim 64, wherein the second protein is detected at a neuromuscular junction.

66. (Previously Presented) The method according to claim 36, wherein the internalization of the fusion protein at the neuromuscular junction is increased.

67. (Previously Presented) The method according to claim 47, wherein the internalization of the fusion protein at the neuromuscular junction is increased.